

**IN THE UNITED STATES PATENT AND TRADEMARK OFFICE**

APPLICANTS: Cauwenberghs *et al.*

CONFIRMATION NO.: 7478

APPLICATION NO.: 10/019,740

GROUP NO.: 1641

FILING DATE: May 8, 2002

EXAMINER: Jung, Unsu

TITLE: Detection of von-Willebrand Factor (vWF) Activity

Mail Stop Amendment  
Commissioner for Patents  
P.O. Box 1450  
Alexandria, VA 22313-1450

**SECOND DECLARATION OF DR. HANS DECKMYN**  
**UNDER 37 C.F.R. § 1.132**

Dear Sir:

I, Dr. Hans Deckmyn, hereby declare and state as follows:

1. I am a recognized expert in the field of von Willebrand's disease and have been working in this field for many years. My *curriculum vitae*, which includes my educational and employment history and a list of my publications and patents, is attached hereto as Exhibit A. My present position is Professor of Chemistry at KU Leuven Campus Kortrijk. I am also a board member of the Belgian Society on Thrombosis and Haemostasis and an executive officer of the European Thrombosis Research Organization.
2. I am a co-inventor of the subject matter claimed in U.S. Patent Application No. 10/019,740, ("the present application"). I understand that the earliest effective filing date of the present application is July 5, 1999. I am also familiar with the November 17, 2008, Office action as well as the Favaloro, Hoylaerts, and Vischer articles, and the Handin U.S. patent cited therein.
3. von Willebrand's disease (vWD) is a common inherited bleeding disorder associated with defects in von Willebrand factor (vWF), a protein required for platelet adhesion. Type 1 and Type 3 vWD are the result of quantitative defects in vWF production. For example,

Type 1 vWD is characterized by reduced plasma concentrations of vWF as compared to normal, while Type 3 vWD is characterized by negligible to absent vWF concentrations. Type 2 vWD is caused by qualitative defects in vWF, *i.e.*, vWF is functionally abnormal. There are 4 known subtypes of type 2 vWD: 2A, 2B, 2M, and 2N.

4. As of the earliest effective filing date of this application, it was understood by those skilled in the art of vWD that high molecular weight (HMW) multimers of vWF are responsible for inducing platelet aggregation. It was understood that such platelet aggregation is induced when HMW multimers of vWF bind to GP1b receptors on platelets. Further it was understood that multimerization of vWF monomers is necessary for bioactivity of vWF.
5. As of the earliest effective filing date of this application, it was understood by those skilled in the art that each of Type 2A vWD and Type 2B vWD are characterized by the absence of HMW multimers of vWF. In Type 2A vWD patients, the absence of HMW vWF multimers is attributable to errors in protein synthesis that interfere with multimer assembly and/or increased sensitivity of vWF to proteases. As a result, Type 2A patients produce only small fragments or monomers of vWF that cannot multimerize. Consequently, the vWF produced by Type 2A patients has a decreased capacity to interact with platelet GP1b receptor.
6. In contrast, as of the earliest effective filing date of this application, Type 2B vWD was known in the art to be characterized by increased affinity of Type 2B vWF for platelet GP1b. This was known to be the result of a genetic defect that increases the binding affinity of Type 2B vWF for the GP1b receptor. As a result, Type 2B vWF binds to platelet GP1b in circulating blood. Upon binding of vWF to the circulating platelets, platelets aggregate and are cleared from the blood. This accounts for the absence of free (unbound to platelet GP1b) high molecular weight vWF multimers in Type 2B patients. In contrast to Type 2B vWF, normal vWF does not spontaneously interact with the GP1b receptor unless vWF is activated as the result of a bleeding injury.
7. If blood, plasma, or serum from a Type 2B patient sample were incubated with ristocetin and isolated GP1b, a skilled artisan, as of the earliest effective filing date, would have expected to detect only low amounts of vWF binding to isolated GP1b. This is due to the

fact that the HMW vWF multimers would already be bound to GP1b receptors on the patient's own platelets. However, some minimal binding from vWF monomers would contribute to the low amount of vWF binding detected.

8. If blood, plasma, or serum from a Type 2A patient sample were incubated with ristocetin and isolated GP1b, a skilled artisan, as of the earliest effective filing date, would have expected to detect only low amounts of vWF binding to isolated GP1b due to the absence of HMW vWF multimers in the patient sample. However, some minimal binding from vWF monomers would contribute to the low amount of vWF binding. The low level of binding detected between Type 2A vWF and GP1b in the presence of ristocetin discussed here in paragraph 8 would be expected to be the same as the low level of binding detected between Type 2B vWF and GP1b in the presence of ristocetin as discussed in paragraph 7.
9. Based on my statements in paragraphs 4-8 above, as of the earliest effective filing date, a skilled artisan assaying Type 2A or Type 2B patient samples in the presence of ristocetin and isolated GP1b would have expected the results from each assay to exhibit the same level of vWF binding, *i.e.*, a low amount, due to the absence of HMW vWF multimers necessary for binding to GP1b.
10. As of the earliest effective filing date, persons of skill in the art were aware that Type 2A vWD and Type 2B vWD could be distinguished from one another because platelets from a Type 2B patient will aggregate in response to low concentrations of ristocetin, while platelets from a Type 2A patient will not. The difference between platelet aggregation patterns between Type 2A and Type 2B are observed only when patient platelets are assayed and not when exogenous platelets, *i.e.*, non-patient platelets, are assayed in the presence of ristocetin. This assay using patient platelets is known as a ristocetin-induced platelet aggregation (RIPA) assay. The aggregation of Type 2B platelets at low concentrations of ristocetin observed in the RIPA assay is a result of the high affinity for platelet GP1b exhibited by vWF from Type 2B patients. Because of the high affinity for platelet GP1b, Type 2B vWF does not require the same level of cofactor assistance from ristocetin to bind to GP1b as required by normal vWF. In contrast, platelets from a Type 2A vWD patient will exhibit no or a little aggregation in the presence of low or high

concentrations of ristocetin because Type 2A vWF contains only small vWF monomers which have a low affinity for GP1b, binding to which is necessary to cause platelet aggregation.

11. Based on my statements in paragraphs 4-10, if ristocetin and an isolated GP1b fragment were used in an assay with a patient sample containing patient platelets (patient platelets being required by a RIPA assay), the isolated GP1b would compete with platelet GP1b of patient platelets for binding to defective Type 2B vWF. Given that some defective Type 2B vWF would bind to platelet GP1b and some to isolated GP1b, this would interfere with the results by reducing the expected amount of agglutination. Further, reduced levels of agglutination would also be seen with patient platelets from a Type 2A vWD patient. Because of the reduced levels of agglutination from the expected levels needed to distinguish between Type 2A and Type 2B vWD, it would not be possible to accurately distinguish type 2A and Type 2B vWD from one another employing such a method.
12. As an inventor of the invention presently claimed in this application, I believe that I and the other inventors were the first to recognize the utility of GP1ba isolated from a platelet, as opposed to using whole platelets, in an assay for detecting vWF ristocetin cofactor activity, that is the binding activity of vWF to GP1ba (or a fragment thereof) in the presence of ristocetin or a functionally equivalent substance.
13. In the process of developing a method for discriminating between different types of von Willebrand disease using a vWF binding activity, I and the other inventors first determined whether the binding activity of normal vWF to soluble forms or portions of GP1ba could be adequately detected in the presence of ristocetin by immobilizing the soluble forms or portions of GP1ba to plastic, for example, to 96 well plastic plates, and would therefore be useful for a clinical assay for detecting vWF binding activity in the presence of ristocetin. We assayed both glycocalicin, a naturally occurring soluble fragment of GP1ba, and a recombinant fragment of GP1ba, GP1ba<sub>(1-289)</sub>, for their ability to bind to vWF in the presence of ristocetin. For each GP1ba fragment immobilized to plastic that we tested, we determined that the binding activity of normal vWF in the presence of ristocetin was negligible and therefore not useful for a clinical assay for

- detecting vWF binding to GP1 $\beta\alpha$  in the presence of ristocetin. We determined that vWF was unable to bind the immobilized GP1 $\beta\alpha$  fragments because immobilization of the GP1 $\beta\alpha$  fragments causes a conformational change in GP1 $\beta\alpha$ , thereby preventing vWF binding to the GP1 $\beta\alpha$  fragments. Accordingly, we concluded that assaying vWF binding activity to GP1 $\beta\alpha$  or a fragment thereof immobilized to plastic in the presence of ristocetin would not be an effective substitute for detecting vWF ristocetin cofactor activity in the presence of platelet-GP1b because the plastic-immobilized GP1 $\beta\alpha$  fragments did not permit detection of any significant vWF binding activity.
14. Given that immobilizing GP1 $\beta\alpha$  fragments was not successful, we sought other means for utilizing GP1 $\beta\alpha$  fragments in a clinical assay for detecting vWF binding activity in the presence of ristocetin. Subsequently, we discovered that in order to detect the binding activity of vWF to GP1 $\beta\alpha$  or a fragment thereof, it is necessary to present the soluble form or portion of GP1 $\beta\alpha$  by an anti-GP1 $\beta\alpha$  antibody. Over 90 monoclonal antibodies against GP1 $\beta\alpha$  were screened in order to detect anti-GP1 $\beta\alpha$  antibodies, that when bound to GP1 $\beta\alpha_{(1-289)}$ , permitted binding of GP1 $\beta\alpha$  to vWF. Over 40 monoclonal anti-GP1 $\beta\alpha$  antibodies were screened in order to detect anti-GP1 $\beta\alpha$  antibodies, that when bound to glycocalicin, permitted binding of glycocalicin to vWF. Anti-GP1 $\beta\alpha$  antibodies were coated on plastic well plates and glycocalicin or a GP1 $\beta\alpha$  fragment were added to the plate. After incubation, normal vWF and ristocetin were added. Binding between vWF and the GP1 $\beta\alpha_{(1-289)}$  fragment or glycocalicin was detected by using an anti-vWF antibody labeled with horseradish peroxidase according to standard ELISA techniques. For 3 anti-GP1 $\beta\alpha$  antibodies, we detected sufficient levels of vWF binding to the GP1 $\beta\alpha$  fragment in the presence of ristocetin. For 1 anti-GP1 $\beta\alpha$  antibody, we detected sufficient levels of vWF binding to glycocalicin in the presence of ristocetin. Accordingly, we determined that these anti-GP1 $\beta\alpha$  antibodies would be suitable for presenting GP1 $\beta\alpha$  in an assay for detecting vWF binding to GP1 $\beta\alpha$  or a fragment thereof in the presence of ristocetin.
15. I further declare that all statements made in this Declaration are of my own knowledge, are true, and that all statements made on information and belief are believed to be true. Moreover, these statements were made with the knowledge that willful false statements

and the like made by me are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Dated: May 16, 2007

By: Hans Deckmyn

Dr. Hans Deckmyn

BGS-1304647 v42

## Curriculum Vitae Hans DECKMYN

**Office address:** Laboratory for Thrombosis Research  
KU Leuven Campus Kortrijk, E. Sabbelaan 53 B-8500 Kortrijk  
(Belgium)  
tel.: 32-56.246.171 (office), .422 (lab), fax: 32-56.246.997  
e-mail: [Hans.Deckmyn@kuleuven-kortrijk.be](mailto:Hans.Deckmyn@kuleuven-kortrijk.be)  
<http://www.kuleuven-kortrijk.be/irc/trombose>

### Professional positions

1977-1980: Doctoral student Laboratory for Biochemistry (G.Préaux)  
1981-1985: Postdoc Center for Thrombosis and Vascular Research, (M. Verstraete, J. Vermeylen)  
1985-1987: Postdoc Hematology-Oncology, Washington U, School Medicine, St.Louis Mo (P.W. Majerus),  
1988-1992: Senior Researcher Center for Molecular & Vascular Biology KU Leuven (D. Collen, J. Vermeylen)  
1992-1996: Associate Professor in Chemistry KU Leuven Campus Kortrijk  
1994-present: Head of the Laboratory for Thrombosis Research, IRC, KU Leuven Campus Kortrijk  
1996-1999: Professor in Chemistry KU Leuven Campus Kortrijk  
1999-present: Full Professor in Chemistry KU Leuven Campus Kortrijk

### Other

1992-1998: Associate Editor "*Thrombosis and Haemostasis*".  
1994-1998: Editorial Advisor "*The Biochemical Journal*".  
96-02,08-09: Member Research Council KU Leuven  
1998-2004: Editor "*The Biochemical Journal*".  
1999-2002: Member Advisory Board "*Thrombosis and Haemostasis*".  
2000-2006: Chairman Interdisciplinary Research Center (IRC), KU Leuven Campus Kortrijk  
2000-2006 Member Board 'Innovation & Incubation Center Kortrijk' (IICK)  
2001-2002 Vice-chairman Advisory Council on Innovation Charter Zuid-West-Vlaanderen  
2001-2006 Member Board 'Researchpark Hoog Kortrijk'  
2002-present Member Advisory Board "*Journal of Thrombosis and Haemostasis*".  
2002-present Member Board 'Belgian Society on Thrombosis and Haemostasis' (BTH)  
2003-present Member Steering committee 'CIS Innovation West-Vlaanderen'  
2004-present Spokesman "Interfacultary Center for Biomacromolecular Structure Research" (BioMacS)  
KU Leuven <http://biomacs.kuleuven.be/index.htm>  
2005-present Board member 'European Cardiovascular Genetics Institute' (ECGI)  
2005-2007 Member Council for Industrial Research KU Leuven  
2006-2008 Section Editor '*Thrombosis and Haemostasis*'  
2006-present Member FWO commission Molecular & Cellular Biology, Genetics

2007-present Executive Officer 'European Thrombosis Research Organisation' (ETRO)  
2007-present Founding member PharmAbs <http://www.pharmabs.org/>

## Various

### Honors and awards

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1978-1979: Fellowship IWONL (Institute for Advancement of Sc.Res. in Industry & Agriculture, Belgium)  
1985: Prize Boehringer-Ingelheim for Research on Thrombosis and Coagulation  
1985-1987: NATO Research Fellowship  
1985-1986: Fulbright Research Award  
1989: Young Investigator Award XIIth Congress on Thrombosis and Haemostasis  
1991 Research Prize "Dr. en Mevr. Schamelhout-Koettlitz"-Foundation for Scientific Research (Royal Academy of Medicine of Belgium)  
1992 Fellowship "Belgian Action against Cancer"  
1993 Triannual Prize Baron Simonart Foundation for Clinical Pharmacological Research  
1995 Research grant O.Dupont Foundation (Royal Academy of Medicine of Belgium)  
1997 Visiting professor "Pro Renovanda" University of Debrecen, Hongarije, Medical School  
2004 Triannual price Sidmar for 'Medical Scientific Research' (Royal Academy for Medicine)  
2009 Doctor Honoris Causa, University of Debrecen, Hungary

### Memberships, meetings, journals

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#### 1. International Society on Thrombosis and Haemostasis (ISTH)

1992-present: Member  
1992-8: Associate Editor Journal of the Society: "Thrombosis and Haemostasis"  
1999-2001: Member Advisory Board "Thrombosis and Haemostasis".  
2002-present: Member Advisory Board "Journal on Thrombosis and Haemostasis".

#### 2. Biochemical Society

1994-present: member  
1994-1998: Editorial Advisor of "The Biochemical Journal"  
1998-2003: Co-Editor of "The Biochemical Journal"

#### 3. European Thrombosis Research Organisation (ETRO)

1995-present: Laboratory for Thrombosis Research, IRC-KULAK elected member  
1997, 2003 Invited speaker ETRO Advanced Teaching Course, Heviz, H; Blankenberge, B

- 2007-10: Executive Officer & webmaster  
4. *Belgian Society on Thrombosis and Haemostasis (BTH)*  
1993-presentmember  
2002-present member of the board & webmaster  
5. *Koninklijke Vlaams Chemische Vereniging*  
1998- present: member

Member Scientific Committee *European Platelet and Granulocyte Immunobiology Symposium*  
(Bamberg, D 1992, Cambridge, UK 1994, Hammeenlinna, SF 1996, S'Agaro, E, 1998,  
Amsterdam,NL, 2000, Lago Maggiore, I, 2002, Rust A, 2004, Tromso, N, 2006, Thun,  
CH, 2008, Beaune, FR 2010)

### Teaching

- 1992-present: Introductory course 'General Chemistry': 1<sup>st</sup> yr Medicine, Biomedical Sciences,  
Biochemistry, Biology, Chemistry, Physics,  
Mathematics, Bio-engineers, Pharmacy  
1992-2003: Chemistry II (Systematics) 1<sup>st</sup> yr Biochemistry, Chemistry, Biology
- 1996-2000: Mechanisms of signal transduction, PhD-training Sci-Bioengin-  
Pharmacy-Biomedic Sciences  
1999-present: Cell Biology (part signal Transduction) : 1<sup>st</sup> yr Medicine, Biomedical Sciences  
2005-present: Biochemistry: 1<sup>st</sup> yr Biochemistry, Chemistry, Biology,  
Bioengineers, Pharmacy  
Since 2003: promotor 7 PhD theses, 5 in preparation

### International Advisory functions

1. 1996-present: Dutch "Nederlandse Hartstichting"
2. 1997, 2001: Austrian "Fonds zur Förderung der wissenschaftlichen Forschung"
3. 1998-present: British "Medical Research Council"
4. 1998: South-African "Medical Research Council"
5. 1999: EU-projects External Expert
6. 1999, 04: Luxembourg "Ministère de la Culture, de l'Enseignement Supérieur et de la Recherche"
7. 2000: Dutch "Nederlandse Organisatie voor Wetenschappelijk Onderzoek"
8. 2003: British "Wellcome Foundation"
9. 2004: Irish "Science Foundation" (SFI)
10. 2004-present French AERES (INSERM)

11. 2004: Irish "Research Funding and Policy Division, Health Research Board (HRB)"
12. 2004, 2007 : Luxembourg "Centre de recherche Public de la Santé"
13. 2006, 2007: French 'Agence Nationale de la Recherche –ANR' Research on rare diseases
14. 2006: Irish "Health Research Board" (HRB)
15. 2006: U of Cambridge UK "Promotions Committee for the Faculty of Biology"
16. 2007-present: French "Agence Nationale de la Recherche–ANR" 'Pathophysiology of Human Diseases'

At present our research group consists of 2 professors, 4 postdocs, 4 PhD-students and 4 technicians.

The yearly budget (incl. salaries) amounts to ~€ 860.000, all from competitive grants.

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## Scientific output

### Publications

- 175 publications (Medline), 4,806 citations, h-index: 33 (ISI)  
of which 1 in Science (26.372), 2 in Circulation (12.755), 24 in Blood ( IF 10.896),  
7 in ATVB (7.221), 5 in J Thromb Haemost (5.947), 8 in J Biol Chem (5.581),
- 14 chapters in books

### Patents

#### **Granted**

1. CELL LINES, LIGANDS AND ANTIBODY FRAGMENTS FOR USE IN PHARMACEUTICAL COMPOSITIONS FOR PREVENTING AND TREATING HAEMOSTASIS DISORDERS

European patent granted 2007/01/31, US patent granted  
Inventors: N. Cauwenberghs, H. Deckmyn

2. "Inhibition of the vWF-collagen interaction by anti-human vWF monoclonal antibody (82D6A3) results in abolition of in vivo arterial thrombus formation in baboons.

European patent granted 4/2008  
Inventors : K. Vanhoorelbeke, N. Cauwenberghs, H. Deckmyn

#### **Pending**

1. "Detection of von-Willebrand factor (vWF) activity"  
EP patent Application, Inventors: N. Cauwenberghs, K. Vanhoorelbeke, H. Deckmyn

### Licenses

A new diagnostic test to determine the quality of von Willebrand Factor, with clearly better reproducibility and sensitivity than the test currently used in the clinic (see patent application and Vanhoorelbeke et al Thromb Haemost. 2000;83:107-13) outlicensed to Instrumentation Laboratories (NJ) and further developed by Biokit (Barcelona). Beta version tested and approved.

**Current research interests**

1. Development of platelet adhesion inhibitors by interfering with the collagen-VWF-platelet GPIb axis as new antithrombotics with a lower bleeding risk, for treatment of e.g. ischemic stroke or TTP
2. Functional genomics in blood platelets (production and characterisation of platelets from genetically manipulated (silencing/overexpressing) hematopoietic stem cells transplanted in irradiated mice)

**Publication list Hans Deckmyn**

1. Defreyn G, Deckmyn H, Vermylen J.  
A thromboxane synthetase inhibitor reorients endoperoxide metabolism in whole blood towards prostacyclin and prostaglandin E2.  
*Thromb. Res.* 26, 389-400, 1982
2. Badenhorst PN, Deckmyn H, Vermylen J.  
The effect of sulphinpyrazone on whole blood thromboxane and prostacyclin generation in man.  
*Thromb. Res.* 28, 59-66, 1982.

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3. Boogaerts MA, Vermylen J, Deckmyn H, Roelant C, Verwilghen RL, Jacobs HS, Moldow CF.  
Enkephalins modify granulocyte-endothelial interactions by stimulating prostacyclin production.  
*Thromb. Haemost.* 50, 572-575, 1983.
4. Deckmyn H, Proesmans W, Vermylen J.  
Prostacyclin production by whole blood from children: impairment in the hemolytic uremic syndrome and excessive formation in chronic renal failure.  
*Thromb. Res.* 30, 13-18, 1983.
5. Spitz B, Deckmyn H, Van Assche FA, Vermylen J.  
Prostacyclin production in whole blood throughout normal pregnancy.  
*Clin. Exp. Hypert.-Hypert. in Pregnancy* B2, 191-202, 1983.
6. Vermylen J, Badenhorst PN, Deckmyn H, Arnout J.  
Normal mechanisms of platelet function.  
*Clin. Haematol.* 12, 107-151, 1983.
7. Vermylen J, Deckmyn H.  
Reorientation of prostaglandin endoperoxide metabolism by a thromboxane synthetase inhibitor: in vitro and clinical observations.  
*Br.J.Clin.Pharmacol.* 15, 17S-22S, 1983.
8. Deckmyn H, Font L, Van Hemelen C, Carreras LO, Defreyn G, Vermylen J.  
Low prostacyclin synthetase activity of fetal rat aorta.  
*Life Sci.* 33, 1491-1497, 1983.
9. Deckmyn H, Van Houtte E, Verstraete M, Vermylen J.  
Manipulation of the local thromboxane and prostacyclin balance in vivo by the antithrombotic compounds dazoxiben, acetylsalicylic acid and nafazatrom.  
*Biochem. Pharmacol.* 32, 2757-2762, 1983.
10. Gresele P, Zoja C, Deckmyn H, Arnout J, Vermylen J, Verstraete M.  
Dipyridamole inhibits platelet aggregation in whole blood.  
*Thromb. Haemost.* 50, 852-856, 1983.

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11. Van Assche FA, Spitz B, Vermylen J, Deckmyn H.  
Preliminary observations on treatment of pregnancy induced hypertension with a thromboxane synthetase inhibitor.  
*Am. J. Obstet. Gynecol.* 148, 216-218, 1984.
12. Boogaerts MA, Van de Broeck J, Deckmyn H, Roelant C, Vermylen J, Verwilghen RL.  
Protective effect of vitamin E on immune triggered granulocyte mediated endothelial injury.

- Thromb. Haemost.* 51, 89-92, 1984.
13. Deckmyn H, Gresele P, Arnout J, Vermylen J.  
BM 13.177 specifically blocks the platelet thromboxane receptor.  
*Arch. Intern. Pharmacodynam. Ther.* 268, 165-166, 1984.
  14. Essien EM, Arnout J, Deckmyn H, Vermylen J, Verstraete M.  
Blood changes and enhanced thromboxane and 6-keto prostaglandin F<sub>1</sub> $\alpha$  production in experimental acute plasmodium bergei infection in hamsters.  
*Thromb. Haemost.* 51, 362-365, 1984.
  15. Gresele P, Deckmyn H, Arnout J, Vermylen J.  
Platelet inhibitory activity of prostacyclin in the presence of erythrocytes as studied with the impedance aggregometer.  
*Br. J. Haemat.* 57, 171-173, 1984.
  16. Gresele P, Deckmyn H, Huybrechts E, Vermylen J.  
Serum albumin enhances the impairment of platelet aggregation with thromboxane synthase inhibition by increasing the formation of prostaglandin D<sub>2</sub>.  
*Biochem. Pharmacol.* 33, 2083-2088, 1984.
  17. Gresele P, Deckmyn H, Arnout J, Lemmens J, Janssens W, Vermylen J.  
BM 13.177, a selective blocker of platelet and vessel wall thromboxane receptors, is active in man.  
*Lancet* i, 991-994, 1984.
  18. Deckmyn H, Gresele P, Arnout J, Todisco A, Vermylen J.  
Prolonging prostacyclin production by nafazatrom and dipyridamole.  
*Lancet* ii, 410-411, 1984.
  19. Spitz B, Deckmyn H, Van Assche FA, Vermylen J.  
Prostacyclin in pregnancy.  
*Eur. J. Obstet. Reprod. Biol.* 18, 303-308, 1984.

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  20. Arnout J, Vanrusselt M, Deckmyn H, Vermylen J, Fiocchi R, Lijnen P, Amery A.  
Platelet hypersensitivity to serotonin after prolonged ketanserin intake?  
*J. Cardiovasc. Pharmacol.* 7 (suppl.7), S20-S22, 1985.
  21. Ceuppens JL, Vertessen S, Deckmyn H, Vermylen J.  
Effects of thromboxane A<sub>2</sub> on lymphocyte proliferation.  
*Cell. Immunol.* 90, 458-463, 1985.
  22. De Maeyer P, Deckmyn H, Arnout J, Vermylen J.  
Intravenous ionic contrast media cause local prostacyclin release in man  
*Investigative Radiol.* 20, 458-463, 1985.
  23. Deckmyn H, Zoja C, Arnout J, Todisco A, D'Hondt J, Vanden Bulcke F, Hendrickx N, Gresele P, Vermylen J.  
Partial isolation and function of the prostacyclin regulating plasma factor.  
*Clin. Sc.* 69, 383-393, 1985.
  24. Gresele P, Arnout J, Deckmyn H, Vermylen J.  
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*Lancet* i, 937-938, 1985.
  25. Gresele P, Bounameaux H, Arnout J, Perez-Requejo JL, Deckmyn H, Vermylen J.

- Thromboxane A<sub>2</sub> and prostacyclin do not modulate the systemic hemodynamic response to cold in humans.  
*J. Lab. Clin. Med.* 106, 534-541, 1985.
26. Janssens WJ, Deckmyn H, Gresele P, Vermeylen J.  
BM 13.177 selectively inhibits endoperoxide analog induced vascular contractions.  
*Arch. Intn'l Pharmacodyn. Ther.* 276, 28-34, 1985.
27. Spitz B, Deckmyn H, Van Bree R, Pijnenborg R, Vermeylen J, Van Assche FA.  
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*Am. J. Obstet. Gynecol.* 151, 116-120, 1985.
28. Van Renterghem Y, Roels L, Lerut T, Gruwez J, Michielsen P, Gresele P, Deckmyn H, Colucci, M, Arnout J, Vermeylen J.  
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*Lancet* i, 999-1002, 1985.
29. Gresele P, Deckmyn H, Arnout J, Zoja C, Vermeylen J.  
Lack of synergism between dazoxiben and dipyridamole following administration to man.  
*Thromb. Res.* 37, 231-236, 1985.
30. Vermeylen J, Deckmyn H, Arnout J, Gresele P.  
A. Schmidt Memorial Lecture: Peroxides in haemostasis and thrombosis.  
*Haemostasis* 15, 8-9, 1985.
31. Vervliet G, Deckmyn H, Carton H, Billiau A.  
Influence of prostaglandin E<sub>2</sub> and indomethacin on interferon-gamma production by cultured peripheral blood leukocytes of multiple sclerosis patients and healthy donors.  
*J. Clin. Immunol.* 5, 102-108, 1985.
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32. Gresele P, Arnout J, Deckmyn H, Vermeylen J.  
Mechanism of the antiplatelet action of dipyridamole in whole blood: modulation of adenosine concentration and activity.  
*Thromb. Haemost.* 55, 12-18, 1986.
33. Vermeylen J, Arnout J, Deckmyn H, Xhonneux B, De Clerck F.  
Continuous inhibition of the platelet S<sub>2</sub>-serotonergic receptors during the long term administration of ketanserin.  
*Thromb. Res.* 42, 721-723, 1986.
34. Vermeylen J, Blockmans D, Spitz B, Deckmyn H.  
Thrombosis and immune disorders.  
*Clin. Haematol.* 15, 393-412, 1986.
35. Gresele P, Arnout J, Coene MC, Deckmyn H, Vermeylen J.  
Leukotriene B<sub>4</sub> production by stimulated whole blood: comparative studies with isolated polymorphonuclear cells.  
*Biochem. Biophys. Res. Commun.* 137, 334-342, 1986.
36. Majerus PW, Connolly TM, Deckmyn H, Ross TS, Bross TE, Ishii H, Bansal V, Wilson DB. The production of phosphoinositide-derived messenger molecules.  
*Science* 234, 1519-1526, 1986.

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